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are necessary for pore formation and translocation. Ident of these residues will aid in elucidating the mechanism of translocation and may be useful in developing therapeutic prophylactic agents against anthrax.

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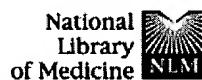
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www.jbc.org**Point mutations in anthrax protective antigen translocation.****Sellman BR, Nassi S, Collier RJ.**

Department of Microbiology and Molecular Genetics, Harvard Medical School, Boston, Massachusetts 02115, USA.

The protective antigen (PA) moiety of anthrax toxin delivers toxin's enzymatic moieties to the cytosol of mammalian cells by a mechanism associated with its ability to heptamerize and form a transmembrane pore. Here we report that mutations in Lys-397, Asp-425, or Phe-427 ablate killing of CHO-K1 cells by a PA ligand. These mutations blocked PA's ability to mediate pore formation and translocation in cells but had no effect on receptor binding, proteolytic activation, or ability to oligomerize or bind the toxin's enzymatic moieties. The mutation-sensitive residues lie in the 2beta(7)-2beta(8) and 2beta(10)-2beta(11) loops of domain 2 and are distant both in primary structure and topography from the 2beta(2)-2beta(3) loop, which is believed to participate in formation of a transmembrane beta-barrel. Our results suggest that Lys-397, Asp-425, and Phe-427 participate in conformational rearrangements of a heptameric pore preformed in the membrane for pore formation and translocation. These

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